"A STUDY ON ANEURYSM OF SINUSES OF VALSALVA – A SINGLE INSTITUTION EXPERIENCE"

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of the requirements for the degree of

M.Ch (CARDIO VASCULAR & THORACIC SURGERY)
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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON ANEURYSM OF SINUSES OF VALSALVA – A SINGLE INSTITUTION EXPERIENCE"** is the bonafide original work of **Dr. S. VIJAY** in partial fulfillment of the requirements for M.Ch., (Cardio Vascular and Thoracic Surgery) Branch-I examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2008. The period of post-graduate study and training was from August 2005 to July 2008.

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DECLARATION

I **Dr. S.VIJAY**, solemnly declare that this dissertation entitled, **"A STUDY ON ANEURYSM OF SINUSES OF VALSALVA – A SINGLE INSTITUTION EXPERIENCE"** is a bonafide work done by me at the Department of Cardio Thoracic & Vascular Surgery, Madras Medical College and Government General Hospital during the period 2005 – 2008 under the guidance and supervision of the Professor and Head of the Department of Cardio Thoracic & Vascular Surgery of Madras Medical College and Government General Hospital, Prof. VARADARAJAN M.S., M.Ch., This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **M. Ch., Degree (Branch-I) in Cardio Thoracic & Vascular Surgery.**

Place : Chennai
Date: **Dr. S. VIJAY**

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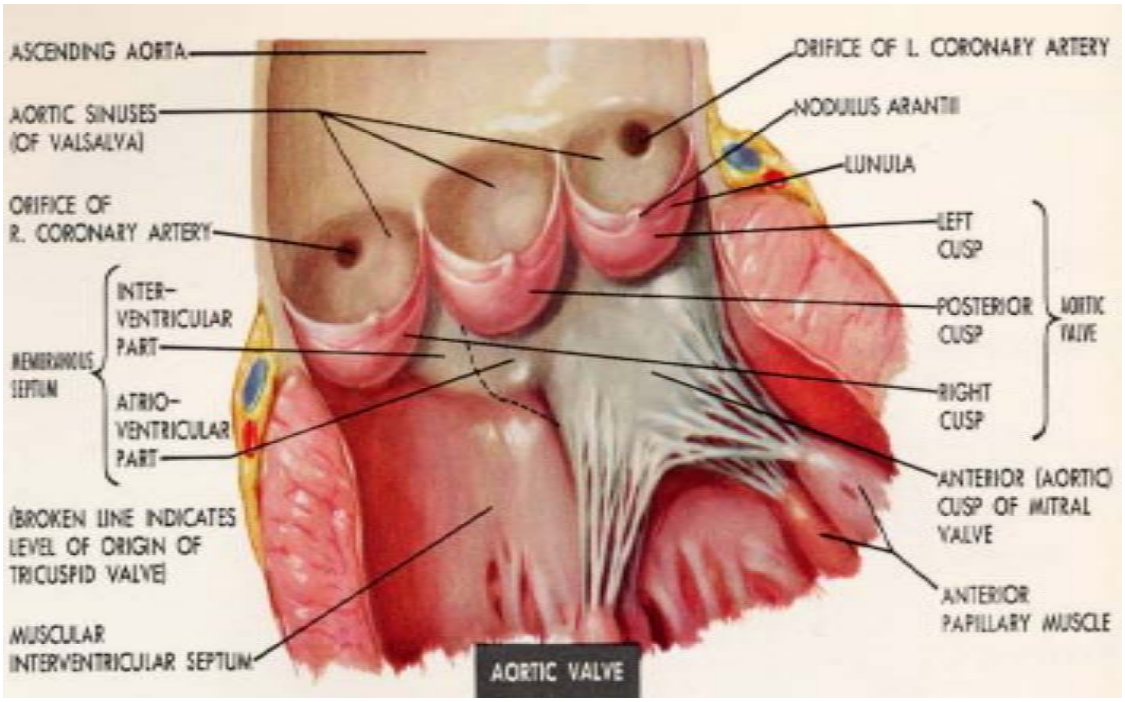
CONTENTS

INTRODUCTION

The sinuses of valsalva are three small outpouchings in the aortic wall just immediately above the attachment of each aortic cusp. Aneurysms of sinuses of valsalva account for 1% of the congenital defects of the heart and circulation.

The aneurysms tend to be single but occasionally more than one sinuses tend to be involved. In 1839, Hope reported a case of aneurysmal pouch of the aorta bursting into the right ventricle. Sakakibara and Konno in their landmark article (1963) noted the prevalence of this lesion in Japan and its association with ventricular septal defects and aortic regurgitation and provided a detailed account of the anatomical aspects of the sinuses of valsalva aneurysms and provided a comprehensive classification of sinus of valsalva aneurysms. Sinus of valsalva aneurysms have protean manifestations.


ASCENDING AORTA
AORTIC SINUSES (OF VALSALVA)
ORIFICE OF R. CORONARY ARTERY
MEMBRANOUS SEPTUM
INTER-VENTRICULAR PART
ATRIO-VENTRICULAR PART
(BROKEN LINE INDICATES LEVEL OF ORIGIN OF TRICUSPID VALVE)
MUSCULAR INTERVENTRICULAR SEPTUM
ORIFICE OF L. CORONARY ARTERY
NODULUS ARANTII
LUNULA
LEFT CUSP
POSTERIOR CUSP
RIGHT CUSP
AORTIC VALVE
ANTERIOR (AORTIC) CUSP OF MITRAL VALVE
ANTERIOR PAPILLARY MUSCLE
AORTIC VALVE

Their presentation may vary from acute asymptomatic murmur to cardiogenic shock and death several operative techniques have been described for aneurysms of sinuses of valsalva.

Subtle modifications are required in the repair of the aneurysms depending on the sinus involved, the presence of coexisting congenital abnormality and the chamber into which the sinus protrudes or ruptures. Most of the patients have a near normal clinical status in the post operative period.

AIMS AND OBJECTIVES

- To study the incidence and demographics of aneurysms of sinuses of valsalva in Indian Population.

- To study the symptomatology of sinus of valsalva aneurysms

- To analyse the incidence of acute symptoms of ruptured sinus of valsalva aneurysms

- To analyse the frequency of involvement of the various sinuses in the aneurysm formation

- To study the incidence of coexisting congenital abnormalities

- To analyse the efficacy of various surgical techniques in the repair of sinuses of valsalva aneurysms with particular reference to the coexisting congenital defects.

- To analyse the clinical status in the post operative period in the operated patients

- To document a couple of interesting cases of sinus of valsalva aneurysms with unusual presentation.

MATERIAL AND METHODS

Between June 2005 and May 2008, 20 patients with aneurysms of sinus of valsalva were operated in the Department of Cardiothoracic Surgery, Government General Hospital, Chennai. We made a retrospective study of these patients reviewing the patient's demographics, clinical status, various associated congenital malformations, the different techniques used in the repair, and postoperative clinical status of the patients.

REVIEW OF LITERATURE

The Sinus of Valsalva is the hollow space enclosed by the three aortic cusps and the aortic wall at the root of the aorta. Sinus of valsalva aneurysm are thin walled saccular or tubular outpouchings usually located in the right sinus or adjacent half of the non coronary sinus and rarely from left coronary sinus. They generally have an intra cardiac course but may protrude into the pericardial space. They may rupture into the heart chambers to form an aorto cardiac fistula or into the pericardial cavity.

The syndrome of acute rupture of a congenital Sinus of valsalva aneurysm was apparently first described by Hope in 1839. A year later Thurman published the first important paper on this subject. Abbot reviewed the clinical feature of acute rupture from eight previous cases and also reported another case. The earlier report of using aortography to diagnose an unruptured aneurysm was that of Falholt and Thomson.

The first successful surgical repairs of sinus of valsalva aneurysm were performed in 1956 at the Mayo clinic and the University of Minnesota using CPB.

Sakakibara and Konno noted the prevalence of this lesion in Japan and its association with VSD and aortic regurgitation and were among the first to provide a comprehensive classification. Their first patient underwent aneurysm repair in 1960.

MODE OF DEVELOPMENT

Several theories were put forward to explain the formation of aneurysm of sinus of valsalva.

1. ***ME Abbot*** in 1919 advanced the opinion that congenital aneurysm of the sinus of valsalva are the result of dilatation caused by the high blood pressure acting on a weak point in the aortic wall due to inadequate fusion of the bulbar septum. Only the right coronary and non coronary sinuses are related to the bulbar septum and when the right and left bulbar ridges do not fuse, a tissue defect occurs which results in the formation of an aortico-cardiac fistula. Even if fusion occurs but is inadequate a point of weakness persists which develops into an aneurysm from the pressure of the blood.

However careful perusal of the literature discloses the aneurysm of the left coronary sinus are not completely absent.

2. ***Vennins*** on 1951 stated that the cause of aneurysm of the Sinus of valsalva was the local defect of elastic tissue at the base of the aorta.

3. ***Edwards*** in 1956 after detailed histologic examination of the base of the aorta stated that the fundamental cause of aneurysm of this area was the separation of the aortic media and the annulis fibrosis of the aortic valve. This defect may result from absence of normal aortic elastic tissue and media in this region. The congenitally weak area gradually enlarges under aortic pressure to form an aneurysm although the age at which this occurs is uncertain. Viewed from the aorta, the aneurysm appears as an excavation of the sinus of valsalva that protrudes into the underlying cardiac chamber.

Precise location of this basic congenital abnormality which may be accompanied by an adjacent separation of the ventricular septum from the aorta to form a VSD tend to be different in Asians and non Asians. In Asians, the basic

abnormality is located left ward and toward the commissural area between the right and left coronary cusps thus compared with non Asians. Rupture occurs more often into the right ventricle than the right atrium.

Also in Asian the coexisting VSD is usually left ward and juxta arterial where in non Asian it is usually right ward and only Juxta aortic. The left ward tendency in Asian's is also manifested by fewer aneurysm of the more right ward non coronary sinus than in non Asian. Left sinus of valsalva aneurysm are uncommon in both Asian and non Asian.

ACQUIRED CAUSES:

Acquired sinus of valsalva aneurysms are caused by medionecrosis, syphilis, atherosclerosis, endocarditis. Behcet's disease or penetrating injuries. They are more readily distinguishable from congenital forms.

They are more diffuse, involving more of the sinuses or multiple sinuses and often the ascending aorta and therefore project into the pericardium outside the heart. A congenital aneurysm is frequently diagnosed by exclusion of other etiologies as well as by presence of associated congenital cardiac defects. Difficulties arise with mycotic aneurysms because endocarditis complicates about 5% to 10% of congenital aneurysm and with medionecrosis because it and marfan syndrome both occur in same patients with congenital sinus of valsalva aneurysm.

RUPTURE:

In some patients the aneurysm gradually develops a localised windsock which ultimately ruptures in an adjacent low pressure cardiac chamber in an unknown percentage of patients. The thin walled ruptured aneurysm characteristically has an intra cardiac fistula portion with a nipple like projection upto the cardiac chamber with one or more points of rupture at its apex. Rarely it projects outside the aortic root or heart.

When the aneurysm coexists with a VSD the windsock usually projects into the right ventricle through a thinned area of myocardium just downstream from the VSD. The aneurysm is separated from the VSD by the hinge line of the aortic valve cusp at the septal portion of the left ventriculoaortic junction.

About one fourth of patient have no windsock or other suggestion of aneurysm formation, but rather have a direct fistulous communication between the aortic sinus and the heart. This defect has been recognised in a few patients at or soon after birth. Windsock deformity is typical in lesion originating from the right sinus and communicating with the right ventricle, a direct fistula is typical in these from the non coronary sinus to the right atrium and an extra cardiac aneurysm is typical in the rare cases of left sinus origin.

CLASSIFICATION OF ANEURYSMS

Type – I: The left part of the right coronary sinus is adjacent to the conus of the right ventricle. Aneurysm frequently originate in this area and protrude just below the comissure of the right and left pulmonary cusps.

Type – II: The central part of the right coronary sinus is next to the crista supraventricularis of the right ventricle. An aneurysm which develops in this area, although quite rare burrows through the crista supraventricularis and protrudes into the outflow tract of the right ventricle.

Type III: The posterior part of the right coronary sinus is separated from the right atrium and ventricle by the membranous septum and is also adjacent to the bifurcation of the vital conduction system.

This area is the second must frequent site of formation of congenital aneurysm. Aneurysm which originate in this area protrude mostly into the right atrium (Type IIIa). A few are found projecting into the right ventricle (Type IIIV) and in rare instances into both atrirum and ventricle.

Type–IV: The right part of the non coronary sinus touches the right atrium. Aneurysm in this area protrude into the right atrium. Because this part of the sinus of Valsalva is near the AV node and bundle of His, aneurysm which originate here may cause arrhythmias.

Aneurysm from the non coronary sinus usually originate from its anterior portion and project into the right atrium. But in rare cases they project and rupture into right ventricle. Rarely rupture can occur simultaneously into the right ventricle and right atrium or into the muscular ventricular septum.

Aneurysm arising from the posterior portion of the noncoronary sinus may rupture into the pericardium. Another rare occurrence is a right sinus or noncoronary sinus aneurysm that rupture into the left ventricle. Rarity of rupture into the left ventricle may be related to the relatively thick wall and high pressure in that chamber. Aneurysms arising from the left coronary sinus may rupture into the left atrium LV or rarely the pulmonary trunk or pericardium. Sinus of valsalva aneurysm rupturing into areas adjacent to the tricuspid valve are also adjacent to the atrio ventricular node and His bundle and may be cause of heart block, bundle branch block, and ventricular fibrillation.

ASSOCIATED CARDIAC ANOMALIES

A VSD is the most common coexisting cardiac anomaly and may arise from the same cogenital anomaly that produce the aneurysm. VSDs occurs in 30% to 50% of patients, but prevalence is higher when the aneurysm arises from the right sinus.

When the aneurysm arises from the left third of the right aortic sinus, the VSD is juxta arterial with its upper margin formed by the confluent aortic and pulmonary valves.

When the aneurysm arises from the central third of the right sinus the VSD may be juxta-aortic or may be within the muscle of the septum's outlet portion.

When the aneurysm arises from the right third of the right sinus the VSD is usually conoventricular and may be perimembranous as well. Rarely a conoventricular VSD occurs

in association with an aneurysm arising from the central or leftward third of the right sinus.

Sakakibara and Konno considered this a coincidental association between two independent malformations rather than a combined developmental anomaly.

AORTIC VALVE ABNORMALITIES AND AORTIC REGURGITATION:

When a VSD is present, AR usually results from a prolapsed aortic cusps similar to the finding in the syndrome of VSD with AR. When a VSD is not present AR usually arises from other aortic valve abnormalities including a bicuspid valve.

PULMONARY STENOSIS:

Important pulmonary stenosis is uncommon in congenital sinus of valsalva aneurysm but small gradients are common.

The stenosis may be valvar but is usually caused by either a projection of the windsock in front of the infundibular septum or a developmental anomaly of the right ventricular outflow tract similar to that present in Tetralogy of fallot and VSD-AR Syndrome.

OTHER ANOMALIES:

Co-arctation of aorta, PDA, ASD, subaortic stenosis and Tetralogy of fallot.

CLINICAL FEATURES AND DIGNOTIC CRITERIA

Unruptured congenital sinus of valsalva aneurysm are usually silent lesions, their diagnosis depends on echo cardiograms or aortograms usually obtained to demonstrate associated symptomatic lesion such as VSD or AR. Diagnosis can be made incidentally during coronary angiography. Rarely unruptured aneurysm produce tricuspid dysfunction or RVOT obstruction bringing the patient to medical care. These aneurysm also produce severe ischemia by compressing the right or left main coronary artery. Embolism from unruptured sinus of valsalva aneurysm has also been reported.

Acute symptoms occur in about 35% of patients with rupture of the aneurysm. In 95% of patients rupture is associated with gradual onset of effort dyspnoea and in 20%, no symptoms develop. Acute symptoms consist of sudden breathlessness and pain. The pain is usually precordial and may

also be epigastric probably because of acute hepatic congestion. Precordial pain may mimic myocardial infarction although radiation of the pain beyond the substernal area is unusual. In few patients, death occurs within days of rupture from right sided heart failure but most patients, improve during the latent period which may last for/weeks months or years. The latent period is followed by recurrence of dyspnea and signs of right sided heart failure.

Characteristic features at this final stage are aortic and tricuspid regurgitation an unusual combination. Acute symptoms at rupture may occur less often with a VSD and more common with AR.

Acute symptomatic ruptures may be precipitated by heavy exertion, but they also occur after serious, automobile accidents and at cardiac catheterisation. Rarely an episode of infective endocarditis may be the precipitating factor.

Marfan syndrome may also predispose the aneurysm to rupture. Rupture is heralded not only by pain and dyspnea but also by a characteristic murmur that is loud, harsh superficial and accompanied by a coarse thrill. The murmur is usually continuous, but it may be to and fro similar to that present in VSD – AR syndrome. In the past this murmur has been mistaken for that of Patent ductus arteriosus but it is maximal at, a lower site usually the left third or fourth ICS.

Rarely the murmur may be systolic when the communication is small. Alternatively the murmur may be diastolic when the rupture occurs into the high pressure L.V. Physical signs include widened aortic pulse pressure, suggesting mild to severe AR. An elevated jugular venous pressure with a prominent V wave, suggesting tricuspid regurgitation, but may also be caused by direct entrance of the fistula into the right atrium, but in most cases this sign is absent until onset of right sided heart failure when liver enlargement and pulsation also occur.

ELECTRO CARDIOGRAM (ECG)

Small slowly developing aortic sinus ruptures are accompanied by normal ECG. The rhythm is normal sinus even when a large rupture is into the right atrium. The PR interval tends to be prolonged Atrioventricular conduction defects including complete heart block and right and left bundle branch block or bifascicular block result when a ruptured or unruptured aneurysm penetrates the base of the ventricular septum and injures the AV node or HIS bundle. The QRS was normal, or rightward, occasionally leftward.

A right atrial P wave abnormality is generated when the right atrium receives the rupture or when an aortic sinus aneurysm causes tricuspid regurgitation increased flow through the left atrium accounts for dilation and for a left atrial or biatrial P wave abnormality.

Rupture into the right atrium or right ventricule results in volume overload of both ventricles but the ECG usually shows LVH by voltage criteria and ST segment and T wave abnormalities. Right ventricular hypertrophy may coexist but does not occur alone and is usually reserved for aneurysm that cause right ventricular outflow obstruction.

THE X-RAY

Because the majority of patients with ruptured congenital aortic sinus aneurysms were previously healthy adults, older routine chest x-rays are often available for comparison. Small insidious perforations leave the x-ray unchanged.

Large acute ruptures are followed by pulmonary venous congestion that initially dominates because of the steep increase in end diastolic Pressure in the unprepared left ventricle, and increased pulmonary arterial blood flow results in enlargement of the pulmonary trunk.

Moderate left atrial enlargement is seen in the lateral projection, a right atrial convexity appears at the right lower cardiac border, and a moderately dilated left ventricle occupies the apex Volume overload of both ventricles with congestive heart failure accounts for the radiologic picture when an aortic sinus aneurysm ruptures into the right side of the heart Rupture

into the left ventricle causes pulmonary venous congestion without increased pulmonary arterial blood flow and with a selective increase in left ventricular size.

Rarely, calcium is deposited in the aortic sinus aneurysm. Also rarely an aneurysm of the Left aortic sinus presents as a localized convex radiologic prominence immediately below the pulmonary trunk, or a large saccular aneurysm of the right aortic sinus presents as a prominent right paracardiac density.

THE ECHO CARDIOGRAM

Echocardiography with color flow imaging and Doppler interrogation establishes the diagnosis of a ruptured or unruptured sinus of Valsalva aneurysm and establishes the presence of associated abnormalities that are intrinsic features of the aneurysm or that are in addition to the aneurysm.

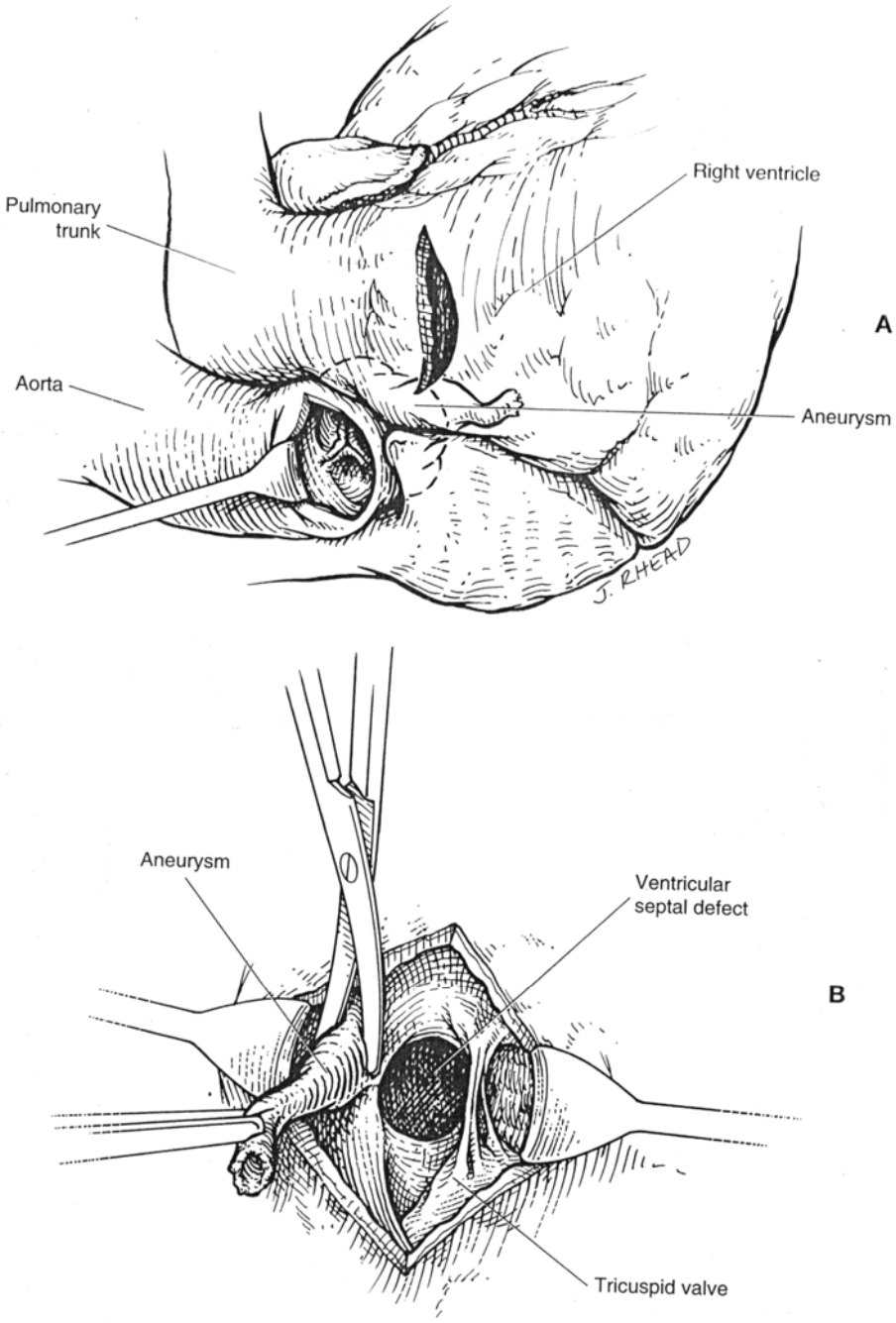
Echocardiography identifies small insidious asymptomatic ruptures suspected only by a continuous murmur Two-dimensional imaging identifies the aneurysmal sac, the aortic sinus of origin, two normal sinuses, and a normal aorta above the aneurysm. Color flow imaging identifies flow into the recipient chamber.

Pulsed Doppler and continuous-wave Doppler define the flow patterns in the ruptured aneurysm. A large, unruptured aortic sinus aneurysm is characterized by phasic expansion and relaxation and to-and-fro pulsed Doppler signals at the site of

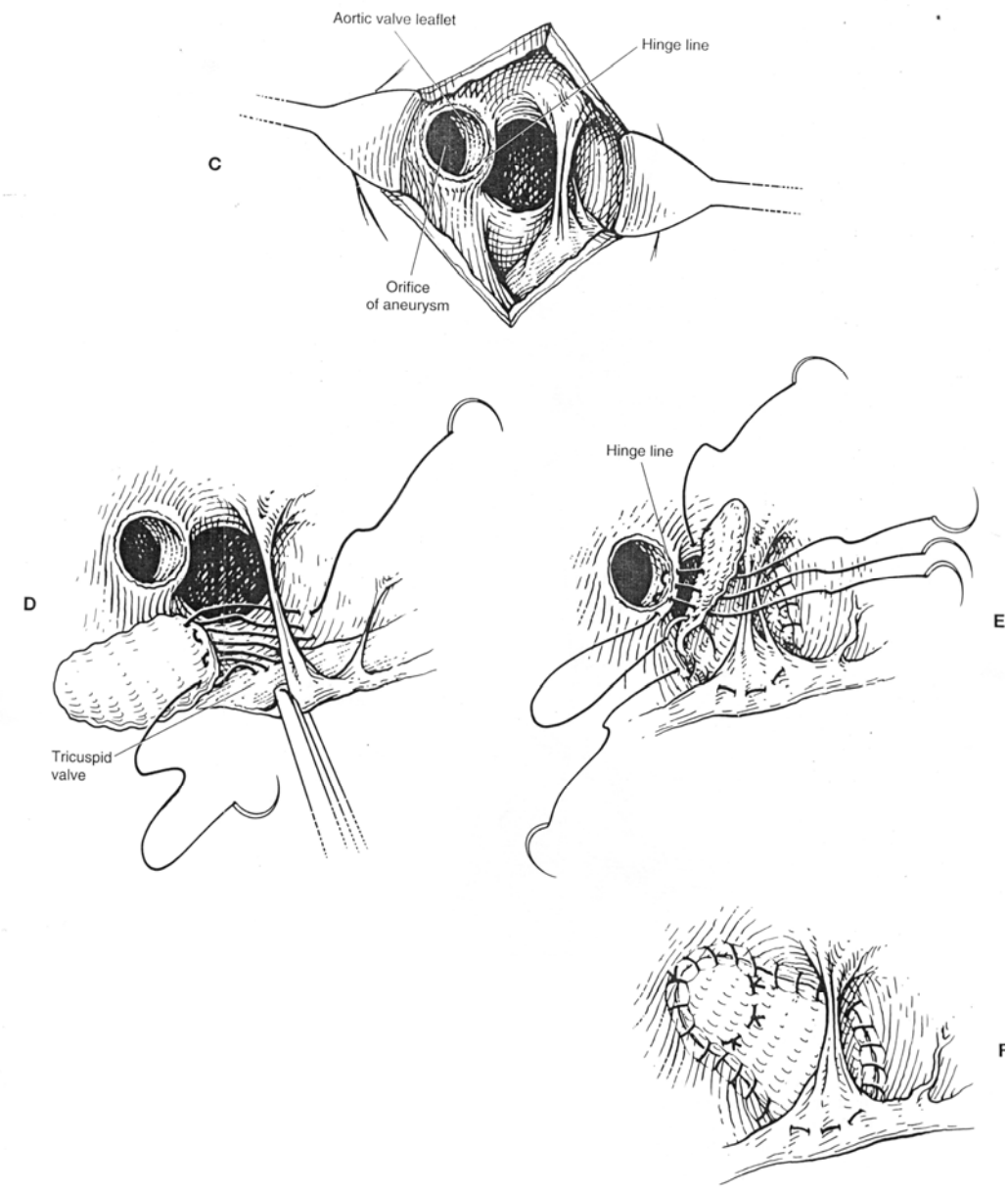
origin from the aorta, but no colour flow evidence of rupture. Doppler interrogation determines the presence and degree of subpulmonary obstruction when an aneurysm protrudes into the right ventricular outflow tract.

The presence and degree of aortic regurgitation are established, a coexisting ventricular septal defect is identified, and the physiologic consequences of rupture into the right atrium, right ventricle, or left ventricle are determined. Real-time imaging identifies ischemic regional left ventricular wall motion abnormalities that result from compression of a coronary artery by an aortic sinus aneurysm.

REPAIR OF RSOV WITH PATCH



REPAIR OF RSOV WITH PATCH



TECHNIQUE OF OPERATION

Ruptured right sinus of valsalva aneurysm with VSD.

If the aneurysm is in the right ward portion of the right sinus, the VSD is probably conventricular and would be approached through the right atrium often with detachment of the anterior and septal leaflets of the tricuspid valve.

If the aneurysm is in the leftward portion of the right sinus of valsalva, the associated VSD in the outlet portion of the ventricular septum would be juxta – arterial and the approach would be through the right ventricle or pulmonary trunk.

In either case operation is usually facilitated by a combined aortic & right ventricular, Pulmonary arterial or a right atrial approach.

After median sternotomy the pericardium is opened and complete external evaluation of the heart is made. The protruding nipple of the ruptured aneurysm may be palpated through the free wall of the right ventricle. Intra operative TEE is useful for defining the location of the aneurysm and the cardiac chamber into which it is ruptured and for assessing the completeness of the fistula and VSD repair and severity of AR before and after repair.

CPB is established after cannulation of the ascending aorta and direct caval cannulation and body temperature is reduced. The aorta is clamped promptly, the right atrium is opened through a short oblique incision and a sump suction catheter is placed across the foramen ovale.

The aortic root is opened transversely and cold cardioplegic solution is infused directly into the left and right coronary ostia or retrogradely through the coronary sinus.

Exposure is obtained by placing stay sutures on the edges of the aortotomy. The orifice of the aneurysm is visualised and elevating the right aortic cusps reveals the underlying VSD. No attempt is made to determine the feasibility of repairing the VSD through the aortic root.

The right ventricle is opened through a transverse or vertical incision depending on distribution of the branches of the right coronary artery. Alternatively an approach can be made through the pulmonary trunk.

The anatomy is visualised. The thinned out wind sock often containing one or more perforations is resected creating a large defect in the right sinus of valsalva. This defect is downstream from the VSD and separated from it by the hinge line of the right aortic cusp. Most of the excised windsock is devoid of aortic media.

A single polyester or pericardial patch is sewn into place to close the VSD and defect in the sinus of valsalva and the area of the hinge line of the right aortic cusp which has been isolated by the resection, is sutured to the patch at an appropriate level.

The ventriculotomy is closed with a continuous prolene stitch and the interior of the Aortic root is again exposed through the aortotomy.

When AR co-exists and the patient is young with pathology limited to prolapse, all or part of a ‘TRUSSLER’ repair of the aortic valve is then performed. In older patients, or when the aortic valve defect is more extensive, valve replacement is necessary.

RSOV WITHOUT VSD:

When the sinus of valsalva aneurysm is usually from the non-coronary sinus but occasionally from the right coronary

sinus ruptures into the right atrium the approach may be through both the aorta and the right atrium. If AR and VSD can be securely excluded the approach may be from the right atrium or aorta alone.

In either situation, CPB is established using direct caval cannulation, an aortic cannula is inserted and the aorta is clamped. The right atrium is opened, obliquely and a sump suction catheter is inserted across the foramen ovale.

A clamp can be placed across the windosock or it can be occluded with a finger. Infusion of the aortic root is begun. If the aortic valve is not completely competent the root infusion is stopped and transverse aortotomy is made and cardioplegic solution is infused directly into the coronary ostia.

Alternatively, cardioplegic solution is administered retrogradely through the coronary sinus.

A co-existing VSD is always sought because it may be over looked during preoperative evaluation, if it is unplugged by a prolapsing aneurysm of the valve cusp. The windsock is then excised remembering the precise location of the hinge line of the valve cusp. When the windsock is narrow and the bordering edges of the sinus are of good quality direct transverse closure of the defect is safe usually however the closure is made with a polyester or pericardial patch.

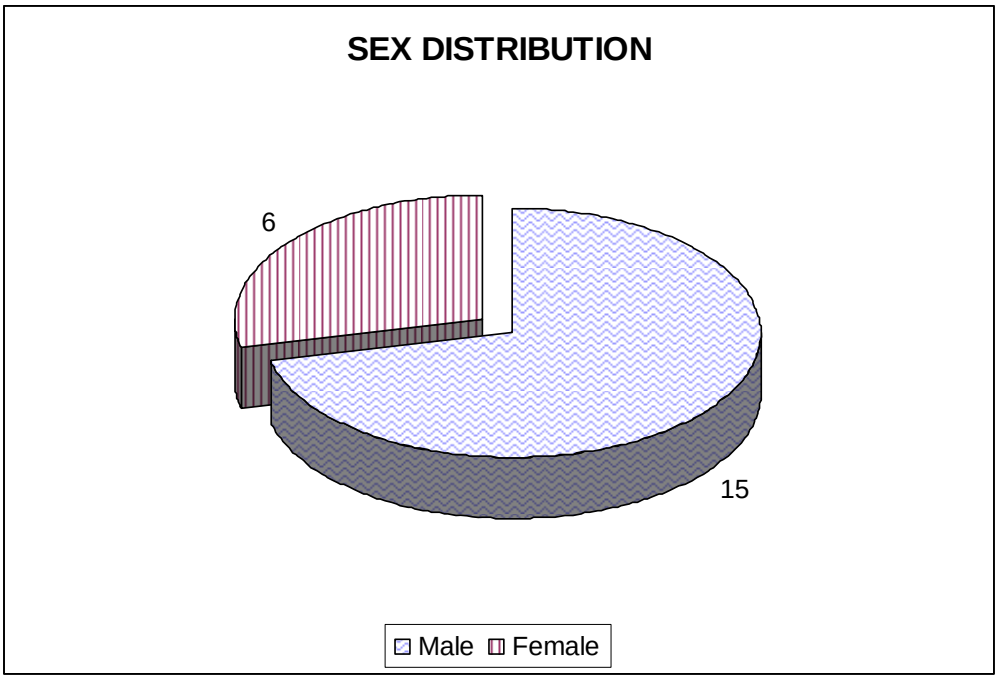
OBSERVATION AND RESULTS

Between June 2005 and June 2008, 21 patients were diagnosed with sinuses of valsalva aneurysm in Government General Hospital, Chennai. 20 patients underwent surgery and 1 patient died Pre-op due to congestive cardiac failure.

SEX DISTRIBUTION:

Fifteen were male patients and 6 were female patients.

Male	*Female*
15	6

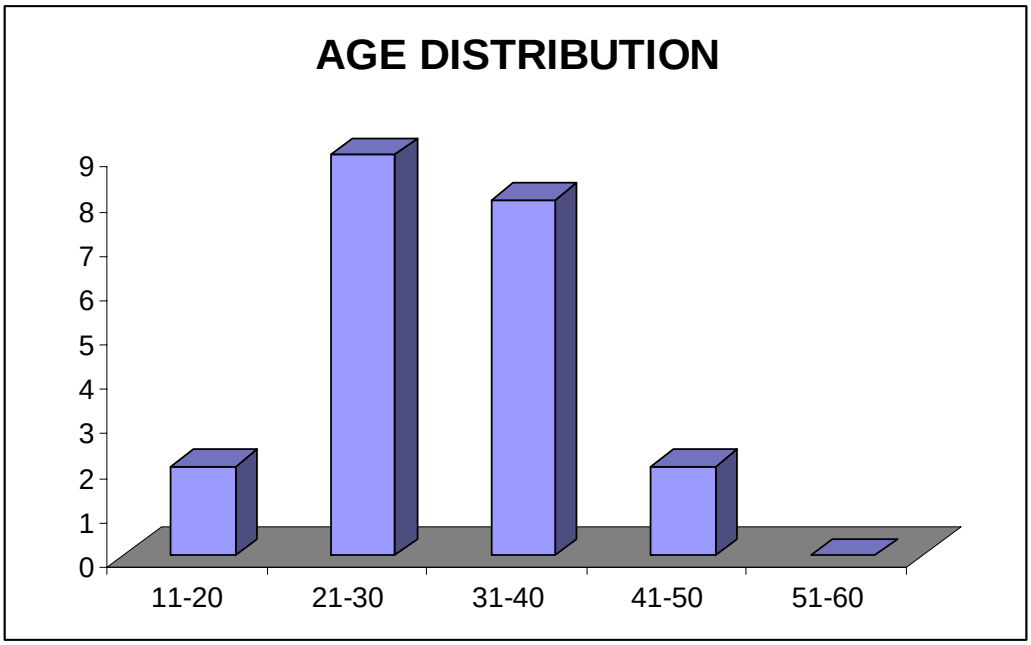


AGE DISTRIBUTION:

Age ranged from 10 years- 45 years. The youngest patient was 11 years old female patient who had a RSOV into RVOT and the oldest patient was a 45 year old male patient who also had a rupture of RSOV → RVOT.

AGE DISTRIBUTION

Age	***Number***
11-20	2
21-30	9
31-40	8
41-50	2
51-60	–

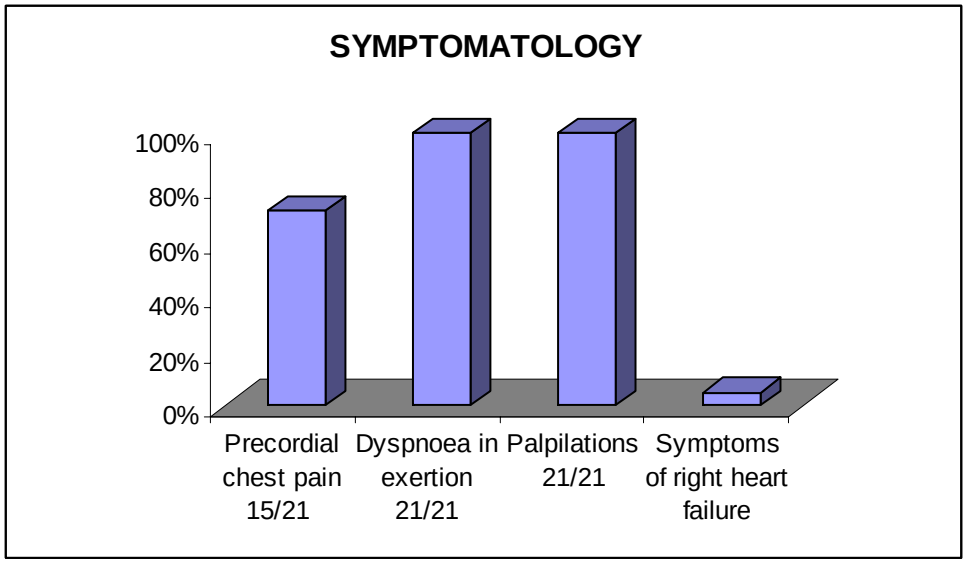


About 9 patients gave history of acute onset of symptoms. The symptoms included acute onset of chest pain and dyspnoea an exertion. Acute symptoms occured during a trivial accident in one patient and after severe exertion in one patient.

Patients with acute onset of symptoms	*Patients without acute symptoms*
9 (Chest pain & Dyspnea)	12

The symptomatology of patients includes

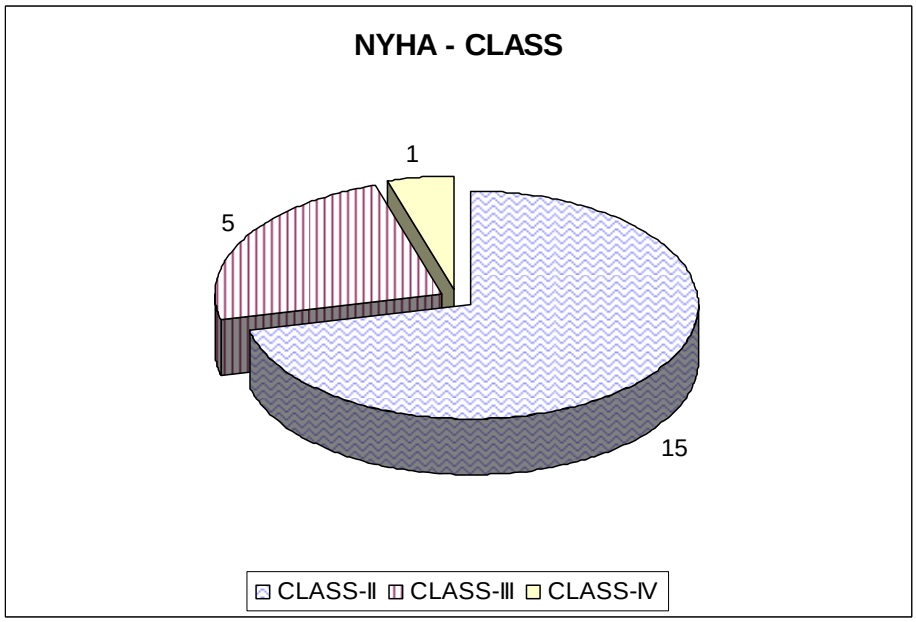
- Precordial chest pain 15/21 : 71.4%
- Dyspnoea in exertion 21/21 : 100%
- Palpitations 21/21 : 100%
- Symptoms of right heart failure: 4.7%



15 Patients were in Class II symptoms.

5 patients were in Class III

1 patient in Class – IV



SIGNS:

All the patients had systolico diastolic murmur except one patient who had only diastolic murmur. One patient had signs of right heart failure also.

Pre operative systemic pulse pressure ranged from 40 to 120mmHg. All patients were diagnosed by transthoracic echo. 6 patients underwent cardiac cathetherisation. One patient had MRA also. This patient had unruptured sinus of valsalva aneurysm burrowing in the inter ventricular septum. All patients were in sinus rhythm.

8 patients had VSD and out of the 8 patients 7 patients were diagnosed, pre operatively as having a VSD and in only one patient additional presence of VSD was diagnosed during surgery.

The VSDs were subarterial in 7 patients one had conoventricular VSD.

AORTIC REGURGITATION:

6 patients had significant AR which required intervention.

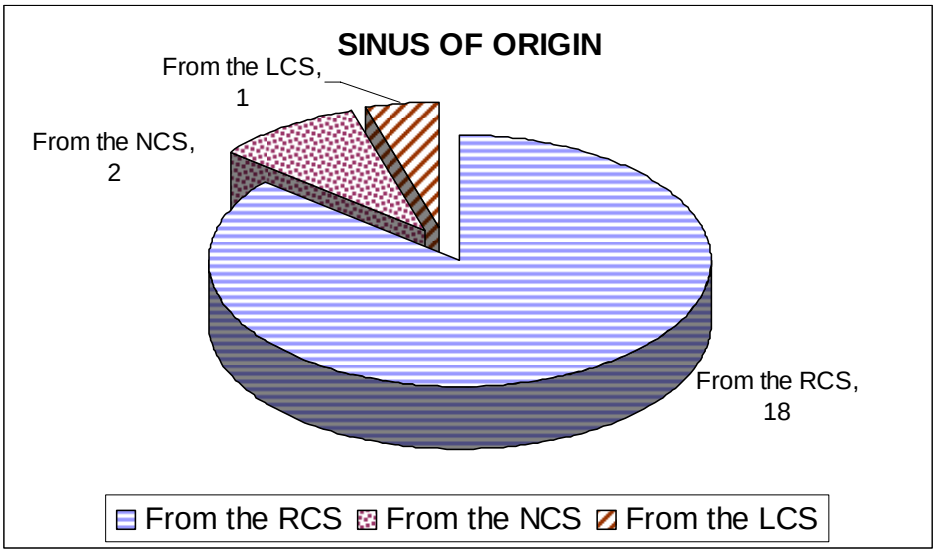
Moderate AR – 5 patients

Severe AR – 1 patient

At operation cardiopulmonary bypass was used with moderate hypothermia 27-32^{0}C. After aortic cross clamping, the aneurysm fistula was visualized through the cardiac chamber receiving fistula blood flow i.,e the right ventricular outflow tract or the right atrium. When the aortic valve was competent and the aneurysm long enough the fistula was clamped before cardioplegia was administered. Otherwise cardioplegia was infused directly into the coronary ostia via an aortotomy. The aortic valve, sinuses of valsalva and coexisting lesion were inspected and repaired.

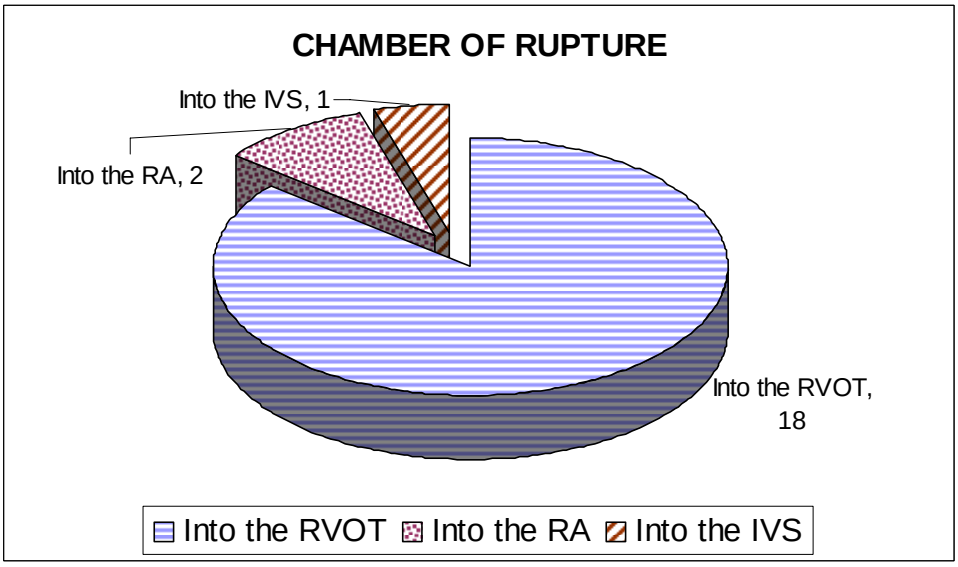
The frequency of ruptured sinus of valsalva origin and exit are as follows:

From the RCS : 18
From the NCS : 2
From the LCS : 1



CHAMBER OF RUPTURE

Into the RVOT : 18
Into the RA : 2
Into the IVS : 1



There was no rupture into the LA or LV or into the pericardial Sac.

Out of the 8 patients with VSD. 7 patients had subarterial VSD and one patient had a conoventricular VSD.

There was no ASD or heart block. No patient had a RSVA in more than one sinus.

One patient had dextrocardia with RSOV.

Aortic valve abnormality was found in 6 patients. All the patients had tricuspid valve.

5 patients had moderate AR (by Echo)

1 patient had severe AR.

Repair of the RSVA fistula was done through an incision in the heart chamber of fistula exit. An aortotomy was made in 6 patients.

RA was opened in 2 patients.

RVOT was opened in 18 patients.

In all the patients after excising the aneurysm the base of the aneurysm was closed with interrupted pledgeted 4-0 prolene sutures. Two patients had a patch closure of the aneurysm of the sinuses of valsalva from inside the aorta. In the patient who had an unruptured aneurysm into the IVS aneurysm opening was closed with a PTFE patch and aortic valve repair was done by TRUSSLER’s technique.

6 patients had direct closure of the VSD. 2 patients had patch closure of the VSD with a PTFE patch.

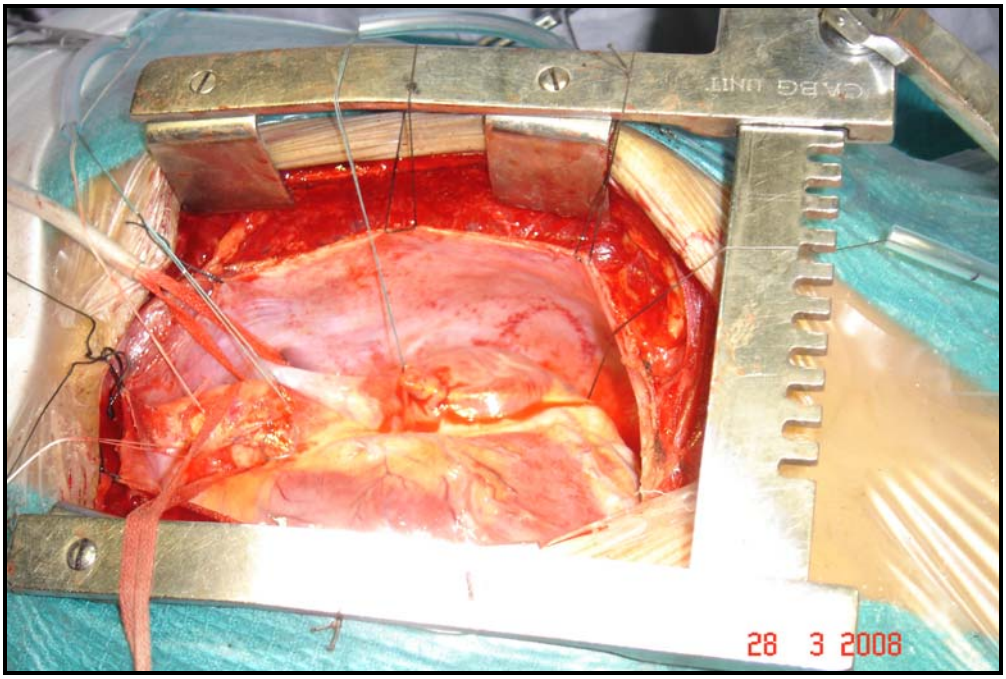
4 patients had aortic valve repair. Repair was by Trunker’s technique were by the redundant RCC was plicated.

2 patients had AVR with 19 mm SJ valve prosthesis.

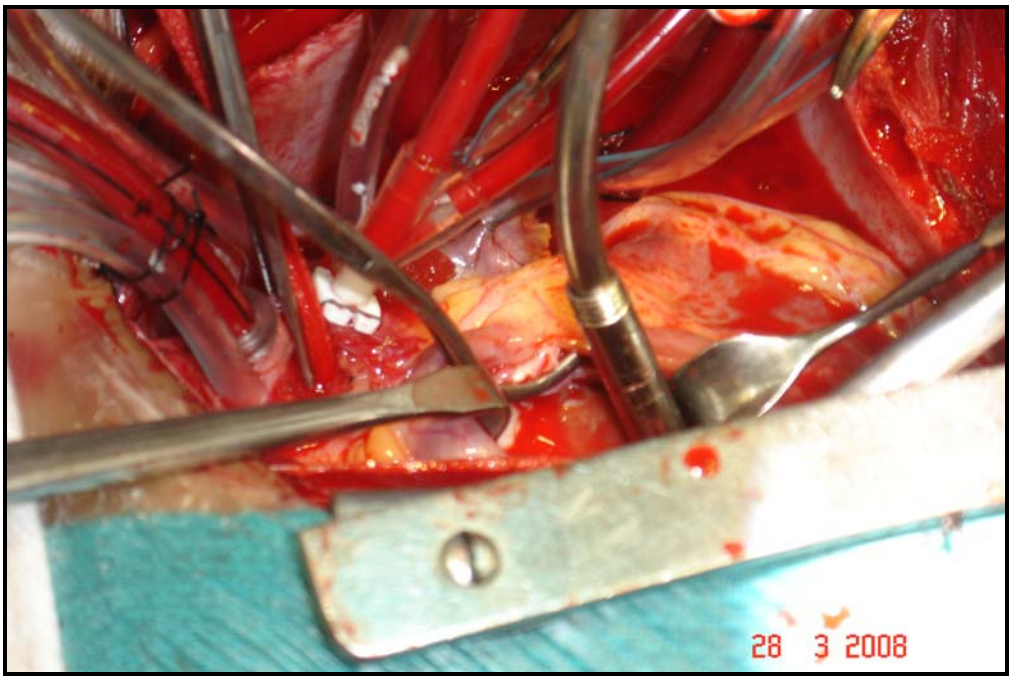
Post operatively all the patients had elective ventilation overnight and were extubated in the next day morning.

12 patients an regular follow up and three patients are in Class I symptoms.

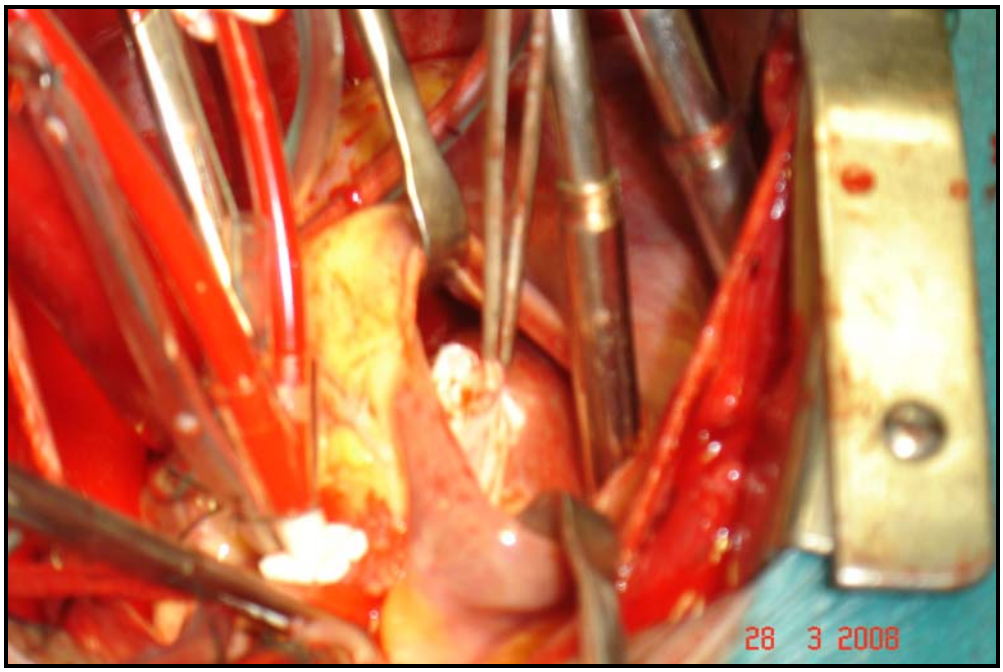
One patient who was in class IV symptoms and had signs of right heart failure died before surgical intervention.



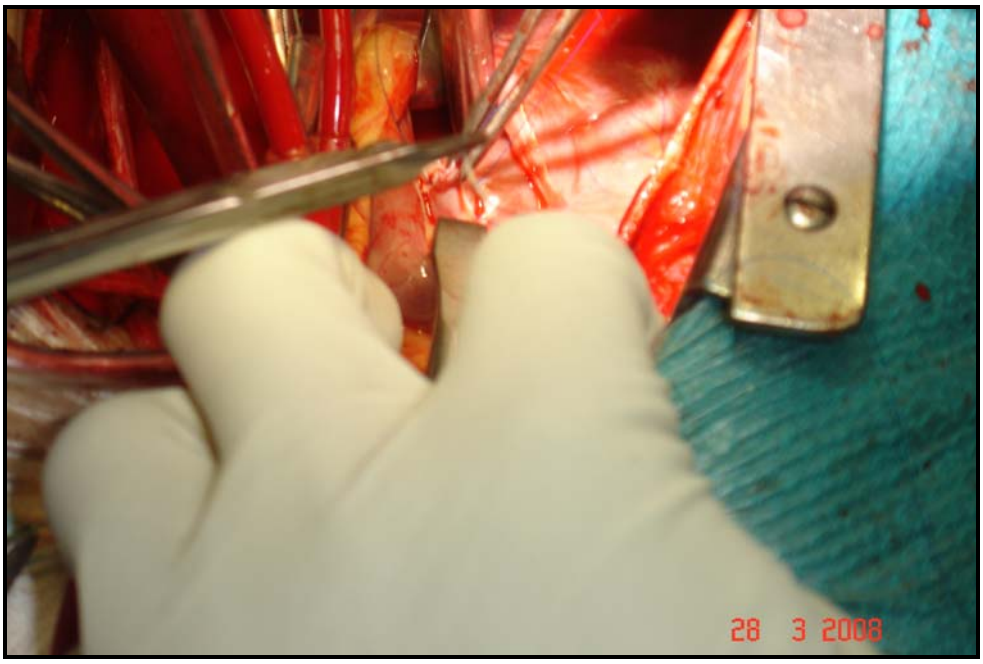
DEXTROCARDIA WITH RSOV



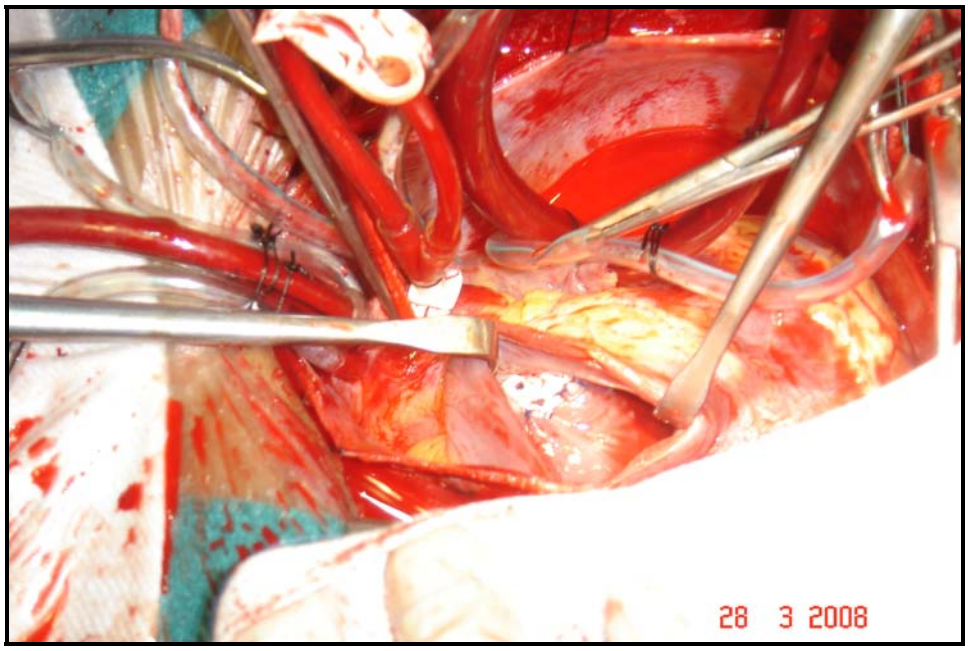
ANEURYSM SAC HELD WITH A CLAMP



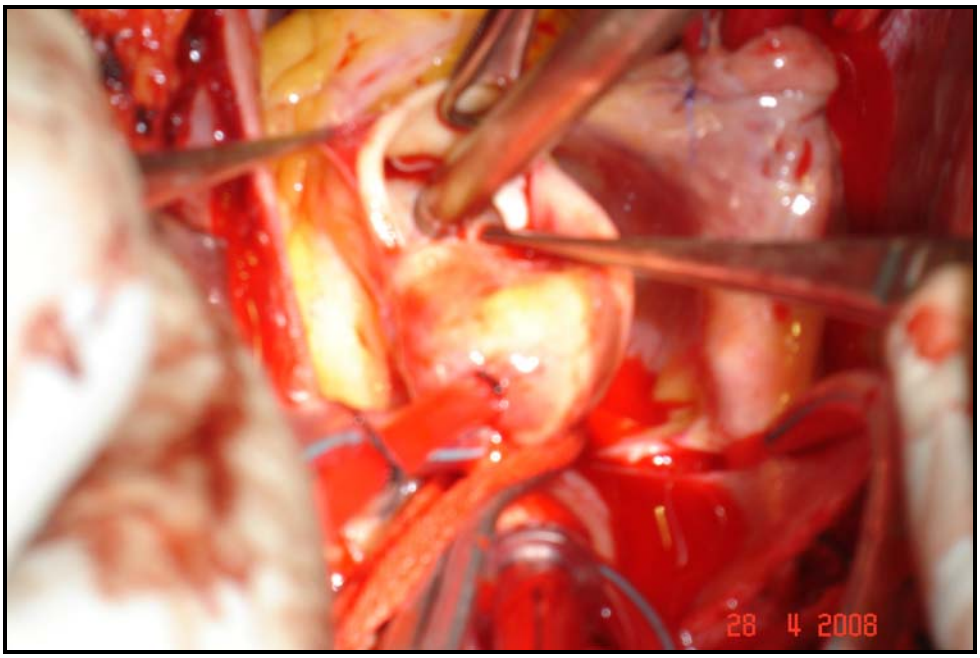
ANEURYSM SAC EXPOSED THROUGH RVOT



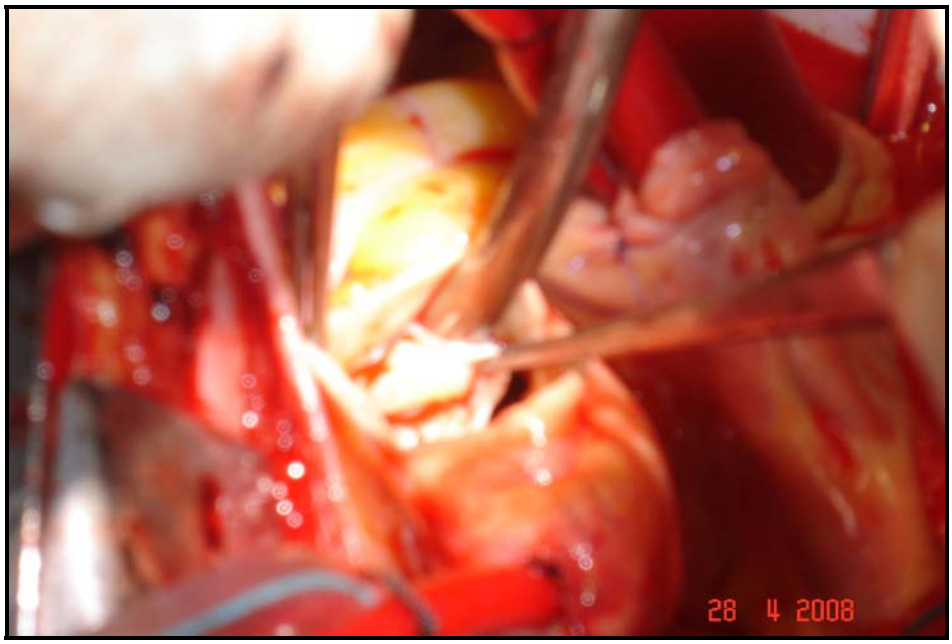
CLOSURE OF FISTULA



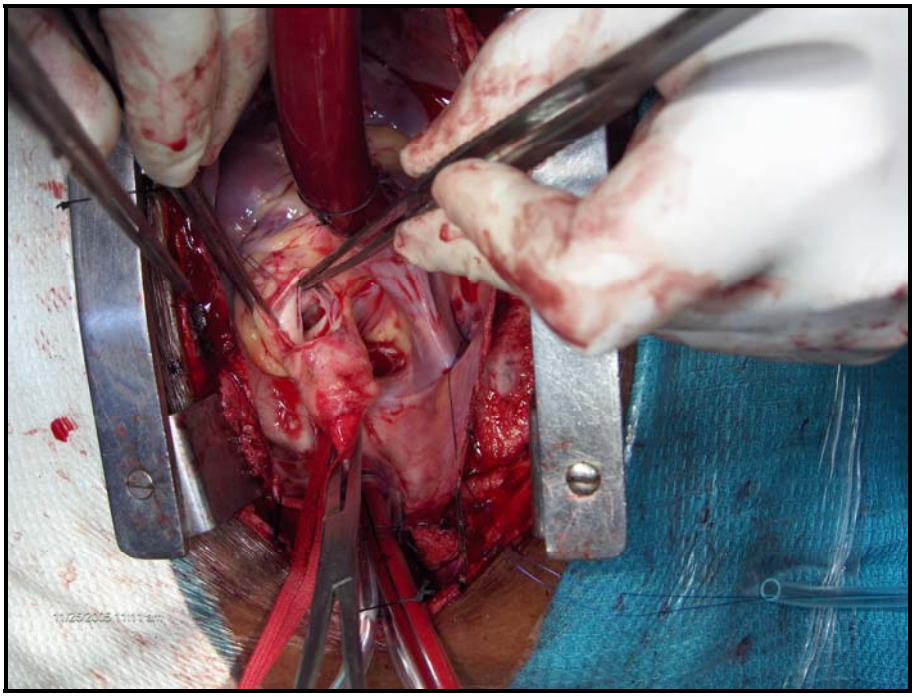
PLEDGETTED CLOSURE OF THE FISTULA



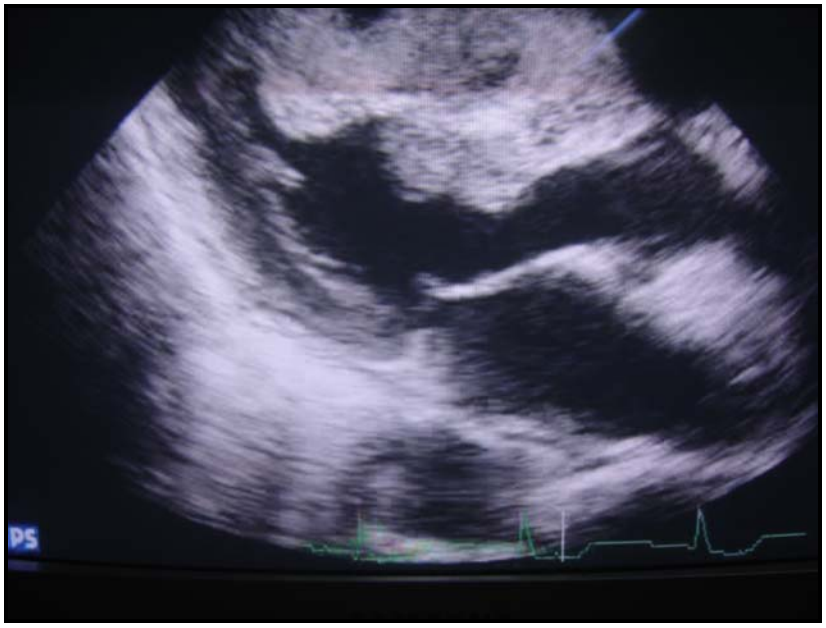
ANEURYSM THROUGH AORTOTOMY



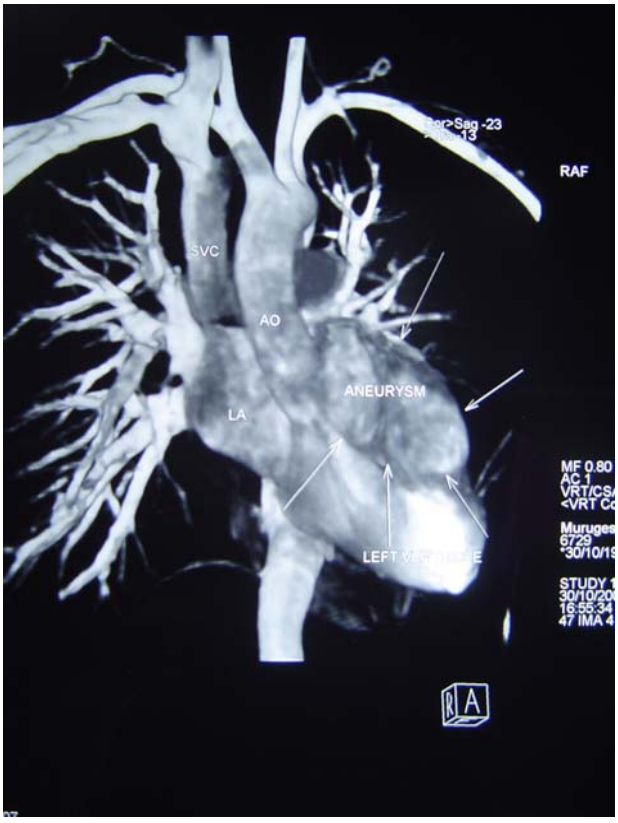
ANEURYSM EXPOSED THROUGH AORTA



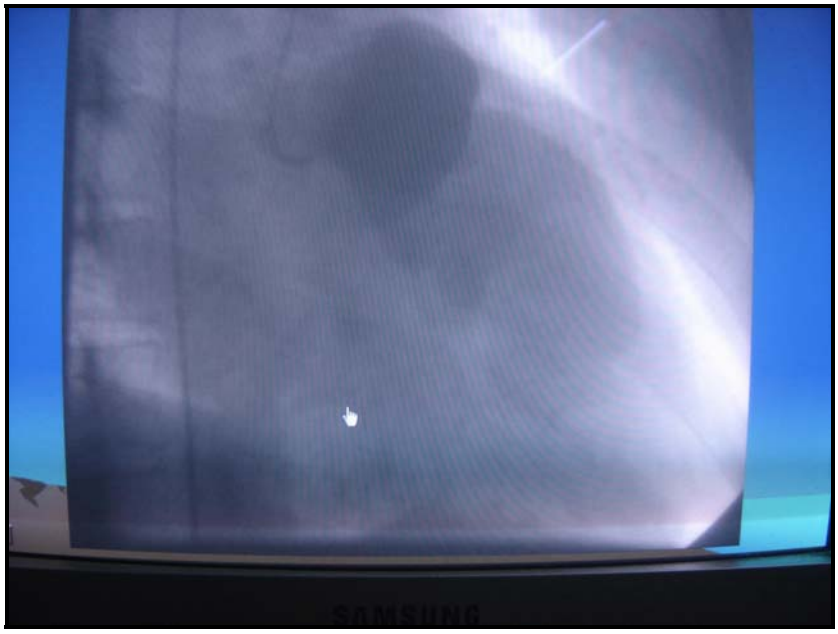
AORTA AND RIGHT ATRIUM OPENED



ECHOCARDIOGRAM



MRA



AORTIC ROOT ANGIOGRAM SHOWING ANEURYSM

RESULTS

Aneurysms of sinus of valsalva occur predominantly in male patients. Literature gives an incidence of 80% in males and 20% in females. In our series the incidence rate was 71.4% and 28.6% in males and females respectively. Chao Dong et al (ATS, 2002), in their series of 67 patients quoted an incidence rate of 65% in males and 35% females.

Sex Distribution	*Our series*	*Literature*	*Chao Dong et al*
Male	71.4%	80	65%
Female	28.6%	20	35%

The age of presentation varied between 10-45 years.

The maximum no. of patients were in the third decade.

	Our Series	***Chao Dong et al***
Age of presentation	10-45 years	2-57 years

The incidence of acute onset of symptoms is about 35%. In the series of Chao Dong et al there was a high incidence of

acute onset of symptoms of about 58%. The incidence of acute onset of symptoms was 42.8% in our series. As only one patient gave an antecedent h/o an accident, rupture of the sinus of valsalva aneurysm can occur without any precipitating factor.

The severity of the symptoms did not correlate with size of the shunt. But patients with co-existent AR had severe symptoms.

According to literature, VSD occured in 30-50%.

The incidence of VSD in our series was 38%.

	Our Series	***Literature***	***Chao Dong et al***
Incidence of VSD	38%	30-50%	47.7%

AI occurred in 28% in our series.

	Our Series	***Chao Dong et al***
Occurrence of AI	28%	17.9%

The commonest sinus of origin of sinus of valsalva aneurysm is RCC. Aneurysm originated from the right

coronary sinus in 85.7% and from non-coronary sinus in 9.5%, from left coronary sinus in 4.7%

Sinus of Origin	***Our Series***	***Chao Dang et al***
RCS	85.7%	77%
NCS	9.5%	23%
LCS	4.7	–

The most common chamber of rupture of the sinus of valsalva aneurysm is right ventricular outflow tract (85%) and 9% of the patients had the rupture of aneurysm into the RA. One patient had an unruptured aneurysm of sinus of valsalva burrowing into IVS.

Chamber of Rupture	***Our Series***	***Chao Dong et al***
RVOT	85.7%	58.2%
RA	9.5%	38.18%
Unruptured (IVS)	4.7%	–
LV	–	3%

Successful repair was achieved by opening the chamber of rupture in all patients.

In 6 patients both the aorta and the cardiac chamber into which aneurysm ruptured were opened.

Successful repair was achieved by direct pledgetted closure of the fistula.

Two patients had closure of the aneurysm from inside the aorta with a patch, which included one patient who had an unruptured aneurysm burrowing into the IVS.

Direct closure of the VSD was done in 6 patients. In 2 patients both the ruptured aneurysm and the VSD was closed with a single patch and hinge line of the aorta was attached to the patch.

Four patients had aortic valve repair.

Two patients had aortic valve replacement.

All patients had successful repair. There was no operative mortality. There was no residual ventricular septal defect or aortic incompetence. Two patients had wound infection, 12 patients who are in regular follow up have good relief of symptoms.

CONCLUSION

RSOV is more common in males.

RSOV presents more commonly in the third decades.

Rupture of the aneurysm may result in the acute onset of symptoms such as chest pain and dyspnea on exertion.

The most common sinus of origin of the aneurysm of sinus of valsalva is the right coronary sinus and the most common chamber into which the aneurysm opens is the right ventricle.

Successful repair of the aneurysm can be done after excision of the redundant sac of the aneurysm followed by direct closure of the defect with pledgetted sutures.

Adequate access to the site of rupture can be achieved by opening the chamber into which the aneurysm ruptures.

Both the VSD and the opening in ruptured aneurysm of sinus of valsalva can be closed by a synthetic patch.

The aortic hinge line at the base of right coronary cusp should be attached to the patch. Thereby preventing residual VSD and aortic incompetence.

Successful repair of the RSOV and the coexisting congenital defects can be achieved with good symptomatic relief.

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